A STUDY ON PERINATAL OUTCOME IN TERM OLIGOHYDRAMNIOS

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BONAFIDE CERTIFICATE

This is to certify that the study entitled **"Perinatal Outcome in term oligohydramnios"** is the bonafide work done by **Dr.S. Amuthambigai**, at the **Institute of Obstetrics and Gynaecology, Government Hospital for Women and Children attached to Madras Medical College, Chennai**, during the period of her Post Graduate study for MD branch II Obstetrics and Gynaecology from 2003 to 2006 under the guidance of PROF. **DR.CYNTHIA ALEXANDER, M.D.,D.G.O.**

This dissertation submitted to **Dr. MGR. Medical University** is in partial fulfillment of the University rules and regulations for the award of **MD Degree in Obstetrics and Gynaecology.**

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CONTENTS

INTRODUCTION

Liquor amnii, a fluid elaborated by amnion a two layered extra embryonic membrane formed by inner ectoderm and outer somatic mesoderm provides fluid medium for the early development of the embryo protecting it from concussion, pressure, dessiccation, reminiscent of the aquatic origin of life.

Adequate amount of amniotic fluid is essential for the normal growth of the fetus for,it cushions against all sorts of trauma and agitations. Its bacteriostatic properties prevents infection and it functions as a primary source of fetal Nutrients.

In Normal Pregancies, the volume of amniotic fluid increases to about one litre at 36 weeks maximum level. Amniotic fluid volume rises progressively during gestation until 36 weeks,the mean amniotic fluid volume relatively consistent in the level of 700-800ml.After 40weeks there is a progressive decline of amniotic fluid volume at a rate of 8 %per week, with amniotic fluid volume averaging about 400ml at 42 wks. The clinical picture of reduced amniotic volume is termed oligohydramnios.

Using amniotic fluid index of less than 5cm the incidence of oligohydramnios was found to be 2.3% after 34 weeks. Oligohydramnios was associated with increased risk of adverse perinatal outcome. The umbilical cord compression during labour is common with oligohydramnios which increases the risk for caesarean delivery for fetal distress and 5 minute apgar score less than 7 **(Chauhan,1999)**

The decrease of amniotic fluid volume is associated with the increased labour induction, still birth, non reassuring fetal heart pattern, meconium aspiration syndrome and neonatal death.
(Casey & Coworkers ,2000)

This present study is undertaken to asses the perinatal outcome in Amniotic fluid index of 5 cm or less (oligohydramnios) in term pregnancies.

AIM OF THE STUDY

To determine the perinatal outcome in term pregnancies with Amniotic fluid index ≤5 cm.

REVIEW OF LITERATURE

AMNIOTIC FLUID:-

Amniotic fluid forms an aquatic pond inside the amniotic cavity surrounding the fetus. This fluid provides several important benefits to the fetus. Amniotic fluid has a number of important roles in embryo / fetal development like.

1. Permitting fetal movement and the development of the musculoskeletal system.
2. Swallowing of amniotic fluid enhances the growth and development of gastrointestinal tract.
3. Amniotic fluid volume maintains amniotic fluid pressure there by reducing the loss of Lung liquid -an essential component to pulmonary development (Nicolini,1989)
4. The ingestion of amniotic fluid provides some fetal nutrition and essential nutrients.

- Protects the fetus from external trauma
- Protects the umbilical cord from compression
- Its constant temperature helps to maintain the embryo's body temperature
- Its bacteriostatic properties reduce the potential for infection.

FORMATION OF THE AMNIOTIC FIUID

The amniotic fluid in the first trimester is derived from embryo's plasma volume. Because embryonic skin is only 4-cell layers thick , there is rapid diffusion across this permeable barrier into the amniotic cavity.

During the embryo-fetal development, three overlapping excretory systems develop- pronephros, mesonephros and meta nephros. The pronephros and mesonephros are nonfunctional in humans. The metanephri begin to develop by 7 weeks menstrual age and are functional by menstrual age 10-11 weeks. Because glomerular filtration precedes tubular function fetal urine is initially relatively hypotonic As the fetus matures , reabsorption of sodium, chloride and water occurs and the excretion of urea and creatinine increases **(Mannie IW, 1980)**

These alterations in fetal renal function, in part, explain the changes that are seen in the composition of amniotic fluid with advancing gestational age.

During the second half of pregnancy, the stratification and cornification of the fetal skin reduces the diffusion of fetal extra cellular fluid into the amniotic cavity. But the fetal skin continues to play a role in amniotic fluid regulation throughout the pregnancy the importance of this pathway is evident in the significantly higher transcutaneous fluid losses of preterm infants.

According to the current concepts of amniotic fluid dynamics **(Seeds, 1980)** there are six routes for fluid entry into and exit from the amniotic sac for the late gestation fetus.

These are :

1. Fetal urine : is the major source of Amniotic fluid after fetal kidney Function which begins at 10-12 weeks of gestation.
2. Fetal swallowing and reabsorption by intestine
3. Intra membranous pathway
4. Secretion from respiratory tract
5. Oral –Nasal secretions
6. Transmembranous pathway

Fetal urine is the major contributor to the volume of amniotic fluid in the latter half of pregnancy of amniotic fluid ion the latter half of pregnancy. It has been estimated that the volume of urine produced per day by human foetus during the latter half of gestation is 35% of its body weight **(Hedrina,1994)**

The fetal swallowing of amniotic fluid is evidenced by the presence of epidermal debris including lanugo hairs in the meconium It has been

estimated that the foetus swallows amniotic fluid which is equivalent to 15% of its Body weight **(Pritchard 1965)**

The pathway for exchange between amniotic fluid and the fetal blood within the fetal surface of placenta is referred to as intramembranous pathway In the pathway water and solutes can move in opposite directions; i.e. osmotic flow of water and diffusion of solutes occur. It has been estimated that 400ml of water is absorbed daily intramembranously from the amniotic fluid during late gestation .

Fetal lungs secrete large volumes of fluid each day and less than 1% of this secreted fluid is needed to expand the lungs with growth the remainder flows out of the lungs and may either enter the amniotic fluid or be swallowed as it exits the trachea. The amniotic fluid from the trachea is the source of surfactants, which are commonly used as an indicator of fetal lung maturity. Only under condition of fetal asphyxia or severe distress do the fetal lungs absorb fluid. A supporting observation is that, although meconium staining of amniotic fluid is common, aspiration of meconium into the lungs of the newborn is relatively uncommon.Lung secretion rates are approximately are 10% of fetal body weight daily **(Harding R 1994)**

Fetal oral and nasal secretions also enter the amniotic fluid, but it has been found to be less than 1% of the body weight per day

(Brace1 R.A.,994)

In transmembranous pathway, exchange occurs between the amniotic fluid and the maternal blood within the uterine wall .This passive exchange is not likely to be significant during the latter half of the gestation. In recent studies the transmembranous fluid of water was found to be as little as 10 ml daily near term under normal conditions

(Brace 1995)

Pathway(Brace,1997)	**Volume ml/day to the fetus**	**To Amniotic fluid**
Fetal swallowing	500-1000	
Oral secretions		25
Secretions from respiratory tract	170	170
Fetal urination		800-1200
In Intramembranous flow across the placenta, umbilical cord and foetus	400	200-500
	-	10
Transmembranous flow		

POTTER FACIES

REGULATION OF AMNIOTIC FLULD VOLUME

Late in gestation when the amniotic fluid volume averages 700-800ml, 1000 ml daily of fluid flows into the amniotic compartment and 1000ml daily leaves the amniotic compartment. Only minor or moderate aberrations in flows during a period of days to weeks could readily lead to oligohydramnios or polyhydramnios

There are no known sensors for AFV, could be part of a control loop to return AFV toward normal whenever it becomes too high or too low. But fetal urine flow, lung liquid secretion and swallowing are known to be regulated. Intramembranous absorption is controlled by factors that regulate the intramembranous permeability and surface area .All of the primary flows into and out of amniotic compartment are regulated and it is the interaction among these flows that provide the regulation of AFV.

Recent studies **(Brace P.A 1997)** suggest that even slight changes in intramembranous permeability can have very large effects on intramembranous flux rates. Any substance e.g. like prostaglandins excreted by fetal kidneys, or released by amnion or chorion, which enter

the amniotic fluid could potentially alter intramembranous permeability and thus lead to alterations in AFV.

Hormonal factors also play a role in amniotic fluid regulation. Intraamniotic injection of prolactin has been shown to reduce the amniotic fluid by 50% by stimulating the transport of water from the fetal to maternal compartment **(Endocrinology 1997).** Cortisol and antidiuretic hormone also affect the permeability of amnion.

CHANGES IN AFV ACROSS GESTATION

AFV changes in pregnancy were studied by **Brace and wolf (1989)** and their observations were:

- Amniotic fluid volume rises progressively during gestation until approximately 32 weeks .

- From 32 weeks to term, the mean AFV is relatively constant in the range of 700-800ml

- After 40 weeks, there is a progressive decline in amniotic fluid volume at a late of 8% per week, with amniotic fluid volume averaging only 400ml at 42wrs

- The variation in 'normal fluid' volume below the mean value is smaller than the upper variation in the third trimester oligohydraminos(defined as the 5^{th} percentile) which is approximately 300ml . Variations in the upper range is almost three fold greater, so that hydraminos (>95^{th} percentile) varies from 1700-1900ml.

OLIGOHYDRAMNIOS

Definition:-

Oligohydramnios is a condition in which the amount of amniotic fluid is decreased below the normal level (Normal level- AFI- 8-15 cm)

Various authors have given definition for oligohydramnios.

Technique	**Definition**	**Redellence**
1. Dye dilution	200ml	Horsager et al(1994)
2. Dye dilution	500ml	Magann et al (1992)
3. 12studies-Direct measurement or dye dilution	318ml	Brace and wolf (1989)
4. Ultrasound	Single vertical pocket <0.5cm	Mercer et al (1984)
5. Ultrasound	Single vertical .pocket <1cm	Manning et al (1981)
6. Ultrasound	Single Vertical pocket <2cm	Manning et al (1990)
7. Ultrasound	Single Vertical pocket <3cm	Halperin et al and crawley et al (1984,1985)
8. Ultrasound	Two diameter pocket (vertical x horizontal)<15cm	Magann et al (1992)

9. Ultrasound	AFI<5th percentile for GA	Moore (1990)
10.Ultrasound	AFI <5 cm	Phelan (1987)
11.Ultrasound	AFI <7cm	Dizon-Townson(1996)
12.Ultrasound	AFI <8cm	Jeng et al (1992)

SONOGRAPIHC ASSESMENT

The sonographic assessment of amniotic fluid volume is semi-quantitative. Historically, the amniotic fluid was simply evaluated visually and graded as decreased, normal or increased. The appearance of fetal crowding and an obvious lack of amniotic fluid were used to define oligohydramnios. **Goldstein and Filly (1988)** have reported good intra-observer and inter-observer agreement between subjective assessment and single largest pocket determination of amniotic fluid volume. One disadvantage of the subjective assessment of amniotic fluid volume is an inability to compare results from serial examinations as the fetal or maternal condition changes.

Manning and platt (1980) measured the single deepest pocket of amniotic fluid free of fetal extremities and umbilical cord to asses amniotic fluids volume .This definition was found to be restrictive. Manning redefined normal amniotic fluid as one packet of amniotic fluid that measures atleast 2cm in two perpendicular planes. **(Manning 1995)**

The amniotic fluid index was proposed on a way to more fully assess the amount of amniotic fluid throughout the uterine cavity **(phelan 1987).** This method summed the maximum vertical pocket of amniotic fluid in each quadrant of the uterus. Oligohydramnios was defined as

amniotic fluid index<5cm. **Moore and Cayle (1990)** obtained AFI's in 791 normal pregnancies They defined oligohydramnios as an AFI below the 5th percentile for gestational age . This value varies between 7.9cm at 16 weeks and 6.3cm at 40 weeks gestation.

Jeng and coworkers (1992) have defined oligohydramnios as an AFI <8cm.

AETIOLOGY OF OLIGOHYDRAMNIOS :

A number of conditions have been found to cause diminished amniotic fluid .They include **(Peipert and Donnenfeld 1991) (Sherer,1990)**

FOETAL

- chromosomal abnormalities
- congenital anomalies
- Growth restriction
- Fetal demise

- Post term pregnancy
- Ruptured membranes

PLACENTAL

- Abruption
- Twin to twin transfusion

MATERNAL

- Uteroplacental insufficiency
- Hypertension
- Preeclampsia
- Diabetes
- Hypovolemia

IATROGENIC

- Drugs like prostaglandin Synthetase inhibitors, angiotensin converting enzyme inhibitors, etc.
- chorionic villus sampling.

IDIOPATHIC

FETAL ANOMALIES AND OLIGOHYDRAMNIOS

Oligohydramnios occurs when there is either obstruction to fetal urinary tract or renal agenesis this occurs due to the anuria.

A number of congenital anomalies have been associated with oligohydramnios.

They are **(Mc curdy and seeds, 1993) (Peipert and Donnenfeid 1991)**

- Amniotic band syndrome
- Cardiac : Tetralogy of Fallot , septal defects
- Central Nervous System : Holoprosencephaly , meningocele, encephalocoele, microcephaly.
- Chromosomal abnormalities : Triploidy, trisomy 18, Turner syndrome.
- Cloacal dysgenesis
- Cystic hygroma
- Diaphragmatic hernia
- Genitourinary :Renal agenesis, renal dysplasia,

Urethral obstruction, bladder exstrophy, Meckel-Gruber syndrome, uretero-pelvic junction, prune-belly syndrome.

- Hypothyroidism
- Skeletal - Sirenomelia, Sacral agenesis, absents radius, facial clefting.
- TRAP (twin reverse arterial perfusion)sequence

- Twin to twin transfusion
- VACTERL (vertebral, anal, Cardiac, tracheo- oesophageal, renal, limb) association

The prevalence of congenital anomalies and aneuploidy varies between 4.5- 37% and 0.4- 4 % respectively **(Nicolaides 1991, Shipp,1996)**

The reduction in amniotic fluid volume makes an assessment of fetal anatomy more difficult. Transvaginal sonography and colour or power Doppler **(De Vore 1995)** can be used to confirm the presence or absence of kidneys and renal arteries, respectively. Early symmetric intrauterine growth restriction and and oligohydramnios should suggest a possible karyotypic abnormality **(Nicodaides, 1986)**

INTRAUTERINE GROWTH RESTRICTION AND OLIGOHYDRAMNIOS :-

Historically, the association between IUGR and oligohydramnios has been attributed to decreased fetal urine production due to deceased uteroplacental perfusion. But,recent animal studies have not found that hypoxemia affects urine formation. It has been proposed that a reversal of

intramembranous flow is responsible for oligohydramnios. There is direct relationship between decreased amniotic fluid volume and the prevalence of IUGR. When a single pocket of amniotic fluid is > 2cm, between 1& 2cm, and <1cm, the prevalence of IUGR is 5% , 20% and 37% respectively **(Chamberlain 1984)**

POSTTERM PREGNANCIES AND IUGR:-

There is an acknowledged relationship between prolonged pregnancies (> 42 wks) and an increase perinatal morbidity and mortality (**Chamberlain 1984; Eden 1987**) over 40yrs ago, a relationship was noted between the diminishing placental function of post maturity and oligohydramnios **(Elliot 1961).** As a result, the ultrasonic assessment of amniotic fluid has been used extensively in the antepartum testing of post date pregnancies.

PRETERM RUPTURE OF MEMBRANES AND OLIGOHYDRAMINOS

Preterm rupture of membranes is defined as rupture of membranes prior to 37 weeks gestation. Spontaneous rupture of the membranes between 24 & 34wks gestation occur in 1.7% pregnancies but account for 20% of perinatal death. Second trimester oligohydramnios has particularly poor prognosis with approximate survival rate 10% **(Shipp 1996)**

IATROGENIC OLIGOHYDRAMNIOS

Drugs like nonsteroidal prostaglandin synthetase inhibitors inhibit renal vascular flow & there by reduce amniotic fluid volume. They also cause closure of ductus ateriosus. Prostaglandin synthetase inhibitors are used in treatment of preterm labour and symptomatic polyhdramnios **(Morales 1989,Kiss how 1990)**.When prostaglandin synthetase inhibitor is discontinued, amniotic fluid gradually reaccumulates. Serial ultrasound examinations are warranted when late second or third trimester women are placed on prostaglandin synthetase inhibitor.

Exposure to angiotensin converting enzyme inhibitors has been with late onset growth restriction and oligohydramnios followed by prolonged and profound neonatal hypotension and anuria. The most severe consequence in renal tubular agenesis,Which causes early onset oligohydramnios, pulmonary hypoplasia & limb contracures and perinatal death **(Barr and Cohen 1991) (Pryde and co-workers 1993) (Schubiger & associates, 1988)**

Oligohydramnios is an acknowledged complication of first trimester chorionic villus sampling and second trimester genetic aminocentesis. If amniotic fluid volume subsequently returns to normal, the neonatal outcome is generally good **(Shipp 1996, Bronshtein 1991)**

FETAL HYPOXIA AND OLIGOHYDRAMINOS

In case of maternal diseases like Chronic hypertension, Connective tissue disorders, severe preeclampsia, and chronic renal disease fatal hypoxia occurs due to uteroplacental insufficiency. The basis for this association has been derived from animal model **(Deutinger, 1987)**

Experimental hypoxia results in a reflex redistribution of fetal cardiac output, that is renal and plumonary flow decrease, hence urinary output and production of fluid by the lung decreases and the amount of amniotic fluid declines.In pregnant women living at 6000 feet, AFI was higher across gestation than in pregnant women at sea level**(Yancey 1994)**

Hypoxia induced suppression of fetal swallowing is thought to be involved in mediating an increase in AFV during long term hypoxia. It is

speculated that oligohydramnios associated with fetal hypoxia is caused by placental dysfunction in addition to the hypoxia.

MATERNAL HYPOVOLEMIA AND OLIGOHYDRAMNIOS:

Acute maternal hypovolemia has been found to be cause of oligohyramnios **(Sherer,1990)**.Studies show that maternal dehydration ,particularly during a period of days is associated with a reduction in amniotic fluid volume .The changes in amniotic fluid volume may be mediated by the changes in intramembranous flow because the water

induced reduction in fetal osmolality would be expected to reduce intramembranous absorption.**(Flack ,1995)**

FETAL EFFECTS OF OLIGOHYDRAMNIOS:

Early onset Oligohydramnios has been associated with many fetal congenital anomalies and poor fetal outcome **(Shenker and Colleagues,1991) (Gaemel and co-workers 1997)** observed that appropriately grown fetus associated with oligohydramnios prior to 37 weeks had a significant three fold increase in preterm birth.

Adhesions between amnion and fetal parts may cause serious deformities including amputation or abnormal positioning of hands and feet. Subjected to pressure from all sides, the fetus assumes a peculiar

appearance-Potter facies (i.e. prominent epicanthal folds a flattened nose and low set ears) and musculoskeletal deformities.

The incidence of pulmonary hypoplasia is found to be higher with oligohydramnios **(mossinger collegues & winn and associates, 2000).** According to **Fox and Badalian (1994)** and **Laura and colleagues (1995)**, there are three possibilities that account for pulmonary hypoplasia.First ,the thoracic compression may prevent chest wall excursion and lung expansion. Second, lack of breathing movements decreases lung inflow.The third and the most widely accepted model involves a failure to

retain amniotic fluid or increased outflow with impaired lung growth and development.

Third trimester oligohydramnios may be responsible for malpresentaion problems, umblical cord compression, concentration of meconium in the liquor and difficult or failed external cephalic Version **(Hofmeyr, 1991).**

Sarno and Co-workers (1989, 1990) reported that AFI of 5 cm or less was associated with five fold increased caesarean delivery rate.

Baron and colleagues (1995) reported a 50 % increase in variable decelerations during labour and a seven fold increased caesarean delivery.

Casey and co-workers showed a 25% increase in non-reassuring fetal heart rate pattern when women with oligohydramnios were compared with normal controls however, the caesarean rate for this increased only from 3to5 percent

MANAGEMENT OF OLIGOHYDRAMNIOS

The obstetric management of oligohydramnios is determined by its etiology. A careful assessment of both the mother and fetus is necessary with primary disease process like placement of a bladder- amniotic shunt for posterior urethral valves, discontinuing a prostaglandin synthetase inhibitor etc.,the amniotic fluid will return to normal.

Intrauterine growth restriction is managed with appropriate antepartum testing and determining the optimal time for delivery.

Antibiotics and corticosteroids may be utilized with preterm premature rupture of membranes at a gestational age < 32 weeks **(Vermillion, 2000)**

Isolated third trimester oligohydramnios is not necessarily associated with poor perinatal outcome **(Meganan, 1999)**

TRANSABDOMINAL AMNIOINFUSION:

Transabdominal amnioinfusion has been attempted for diagnostic and therapeutic purpose in women with second trimester oligohydramnios **(Quetel, 1992)**

By instilling 40 to 600ml of Normal saline transabdominally, improved visualization in ultrasound was made. Also, by mixing indigo carmine and detection of the dye vaginally a diagnosis of preterm premature rupture of membranes was also made **(Fisk 1991)**. **Miyazaki and Nevaserz 1985** noted that various decelerations due to cord compression are reduced by transabdominal amniofusion.

TRANSVAGINAL AMINOINFUSION:

Three predominant indication for this are meconium stained amniotic fluid, variable decelerations and prophylactically for oligohydramnios. The role of amnioinfusion in variable decelerations or prophylactically for oligohydramnios is not clear but multiple randomized studies show that amnioinfusion is associated with significant decrease in meconium aspiration and meconium aspiration syndrome**(Dye,1994)**

MATERNAL HYDRATION:

Intravenous hydration with 6500ml of an isotonic increased amniotic fluid volume in markedly dehydrated women **(Sherer, 1990)**

Brace (1989) showed that changes in maternal intravascular volume can alter fetal urine output amniotic fluid volume and to a lesser extent intravascular volume.

Kilpatric & co-workers (1991) oral maternal hydration with 2 liters of water was associated with an increase in AFI by approximately 30%in women with decrease AFI & Normal AFI

MATERIALS AND METHODS

A prospective study on the perinatal outcome in term pregnancies with Amniotic fluid index ≤ 5 cm & control group amniotic fluid index >5cms was carried out in **Institute of Obstetrics & Gynaecology, Government Hospital for Women & Children**, attached to **Madras Medical College, Chennai – 8,** during the period of February 2004 to January 2006.

INCLUSION CRITERIA:

Pregnant women with gestational age more than 37 weeks and Amniotic fluid index 5 cm or less.

EXCLUSION CRITERIA:-

1. Patients with amniotic membrane rupture or draining P/V.
2. Patients with fetus having congenital anomalies like renal agencies, polycystic kidneys.
3. Patients with multiple gestation.
4. Patients less than 37 weeks.

SAMPLE SIZE:

About 150 cases in AFI ≤ 5cms (Study Group)

And 150 Cases in Control Group (AFI > 5cms)

History regarding age, parity, duration of gestation menstrual history, obstetric history and history of any complications in present pregnancy were noted.

General clinical examination was done. Pulse rate, Blood pressure & temperature noted. Symphysiofundal height was measured. Uterine size, presentation & adequacy of amniotic fluid clinically, were noted. Fetal heart rate was counted. Speculum & pervaginal examination was done to rule out draining P/V and confirm the membranes.

Necessary investigations done. A Non stress test was done.

An Ultrasound examination was done for fetal well being and amniotic fluid index was measured by the technique described by **Phelan et al (1987).**

A , curvilinear transducer is used.

By marking, the uterus is divided into four quadrants using the maternal sagittal midline vertically and an arbitary transverse line

approximately half way between the symphysis pubis and upper edge of uterine fundus.

The transducer was kept parallel to the maternal sagittal plane and perpendicular to the maternal coronal plane throughout.

The deepest, unobstructed and clear pocket of amniotic fluid visualized and image was frozen. The ultrasound calipers are manipulated

in such a way to measure the pocket in a strictly vertical direction. The process is repeated in each of the four quadrants and pocket measurement are summed to give the AFI. Patients are grouped according to their AFI, as AFI 5 cm or less and control group > 5cms. The patients are followed up by observing the mode of delivery, if delivery is made by caesarean section, the indication are recorded. The condition of babies is assessed by birth weight, apgar score, color of liquor and the need for neonatal admission. These babies were followed till 28 days after birth.

OBSERVATION

The present study is under taken to study the outcome of term pregnancy with amniotic fluid index 5 cm or less (study group) and control group >5 cm

Total numbers of patients selected were 150 cases in each group.

AGE DISTRIBUTIONS OF PATIENTS

	AFI ≤5cm		Control group	
AGE	**No**	**%**	**No**	**%**
< 20yrs	10	7	6	4
20 - 30yrs	131	87	139	93
> 30yrs	9	6	5	3
Total	150	100	150	100

$X^2 = 2.38$ P = 0.30

It can be found from the above table that

Majority of cases are in the age group of 20 to 30 Yrs

DISTRIBUTION OF CASES WITH PARITY

Parity	**AFI ≤ 5cm**		**Control group**	
	NO	**%**	**NO**	**%**
PRIMI	83	55	85	57
MULTI	67	45	65	43
TOTAL	150	100	150	100

$X^2 = 0.05$ $P = 0.81$

From the above table,

In AFI ≤ 5 - 55% were primi and 45% were multi.

In control group, 57% were primi and 43% were multi. But the difference was found to be non-significant.

MODE OF DELIVERY

MODE	AFI ≤ 5cm		Control group	
	No	%	No	%
VAGINAL	55	37	94	63
Primary LSCS	74	78	34	61
RPT	21	22	22	39
	95	63	56	37
TOTAL	**150**	**100**	**150**	**100**

$X^2 = 20.28$ P = 0.001 OR = 2.90

In AFI ≤ 5cm, 37% had vaginal delivery and 63% had LSCS. 22% were repeat CS.

In control group, 63% had vaginal delivery and 37% had LSCS. 39% were repeat CS.

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INDICATION FOR CAESAREAN SECTION

Indication For caesarean Section	AFI ≤ 5cm		Control group		Total	
	No	%	No	%		
Fetal distress	48	51	16	29	64	Z=4.29 P=0.05
CPD	8	8	15	27	23	
IUGR	11	12	5	9	16	
Failed Induction	10	11	8	14	18	
Breech	13	13	6	10	19	
Others	5	5	6	11	11	
Total	95	100	56	100	151	

From the above table,

In AFI ≤5cm 51%of patients under went LSCS for fetal distress.

In control group 29% of patients under went LSCS for fetal distress.

The difference was found to be significant.(P=0.05)

INDUCTION OF LABOUR.

	AFI ≤5cm		Control group	
	NO	%	NO	%
No.of Induction	48	42	38	25
Vaginal delivery	12	25	26	68
LSCS	36	75	12	32

$X^2 = 16.21$ P = 0.001 CI (2.3-19)

Induction of labour is 42% vs 25% in AFI < or =5cm and control group.

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COLOUR OF LIQUOR

Liquor colour	AFI ≤5cm		Control group		Total
	No	%	No	%	
Clear	82	54	112	75	194
Thin	30	20	27	18	57
Thick	38	26	11	7	49

$X^2 = 1.19$ P = 0.17

26% of patients ,in AFI ≤ 5 had thick meconium and only 7% of patients in control group had thick meconium

The difference was found to be non significant. (P = 0.17)

BIRTH WEIGHT

Birth weight	AFI≤ 5cm		Control group		Total
	No	%	No	%	
>3kg	16	11	29	19	45
2.5-3kg	72	48	83	55	155
2-2.5	24	16	25	17	49
<2	28	18	8	5	36

$X^2 = 15.6$ P = 0.001

About 18% of babies in AFI≤5cm are below 2kg only 5% of babies in control group are below 2kg. The difference was found to be significant (P = 0.001)

APGAR SCORE

Apgar @ 5 Minutes	AFI 5cm or Less No	%	Control Group No	%
≤ 4	5	3	1	0.6
<7	16	11	2	2
>7	134	88	148	98

$X^2 = 9.99$ P = 0.001 OR = 8.8 (2-56)

In study group 11% had apgar less than 7@ 5 minutes and in control group 2% had apgar less than 7@5 minutes.The difference was found to be significant.(P=0.001)

NEONATAL OUTCOME

NST	AFI ≤5cm		Control group		
	No	%	No	%	
REACTIVE	101	67	126	84	
Neonatal Death	2	2	-	-	X^2=9.3
NON-REACTIVE	49	33	24	16	P=0.02 OR=8(2-40)
Neonatal death	7	14	1	-	
Total Neonatal death	9	6	1	0.3	Z=2.29 P=0.02

In AFI≤5cm about 67% of patients had reactive NST and 33% had non reactive NST.

In control group,about 84%of patients had reactive NST and 16% had non reactive NST. The difference was found to be significant. (P=0.002)

NEONATAL OUTCOME

	AFI≤5cm		Control group		
	No	%	No	%	
NICU admission	69	46%	25	17%	Z=5.28 P=0.001
Discharged	60	87%	24	96%	
Preterm	10	7%	4	3%	
IUGR	39	26%	10	7%	
Neonatal death	9	6%	1	0.3%	

In AFI < or = 5 cm,46% of babies had NICU admission, 87% of babies were discharged. 6% of neonatal death.

In control group 17% 0f babies had NICU admission 96% of babies were discharged. 0.3% neonatal death. The difference was found to be significant.

PERI NATAL OUTCOME IN AFI < 5cm

	Reactive NST		**Non Reactive NST**		
	No	**%**	**No**	**%**	
Mode of delivery					
Vaginal	45	45	10	20	X^2= 8.28 P=0.004
LSCS	56	55	39	80	OR=3.1 (1-7.6)
Apgar @ 5 min 1. Less than 7	5	5	11	22	X^2=10.6 P=0.001
2. > 7	96	95	38	78	OR=5.6 (1.6-20)
NICU admission	37	37	32	65	Z=3.05 P=0.002 CI=28%
Neonatal Death	2	2	7	14	Z=1.82 P=0.02 CI=9%

In study group reactive NST is compared with NR NST.

1. In Reactive NST 45% has vaginal, 55% had LSCS

   Non-reactive NST 20% had vaginal 80% had LSCS

   The difference was found to be significant (P=0.004)

2. In Reactive NST 5% had Apgar less than 7@ 5 min Vs 22% in NR NST. The difference was found to be significant (P=0.001).

3. In Reactive NST 37% had NICU admission, about 65% in NR NST group had NICU admission. The difference was found to be significant. (P=0.002)

4. In Reactive NST 2% had Neonatal Death. 14% of Non reactive NST had Neonatal. Death. The difference was found to be significant. (P=0.02).

CONTROL GROUP

	Reactive NST		Non Reactive NST		
	No	**%**	**No**	**%**	
Mode of delivery Vaginal	86	68	8	33	$X^2 = 10.51$ P = 0.001
LSCS	40	32	16	67	
Apgar @ 5 min 1.Less than 7	1	0.8	2	8	$X^2 = 5.85$ P = 0.02
2.> 7	125	99.2	22	91	
NICU admission	16	13	9	38	
Neonatal Death	-	-	1	4	Z = 2.58 P = 0.10

The risk of apgar less than 7 @ 5 min is 8% in NRNST and 0.8 in reactive NST

DISCUSSION

Oligohydramnios AFI less than or equal to 5cm is associated with increased perinatal morbidity and mortality. With Oligohydramnios meconium, Fetal heart rate abnormalities and low apgar scores are more frequent. Neonatal & Fetal acidosis rates were doubled compared with controls **(Moore et al 1997) fetal** distress requiring operative intervention was tripled compared with control.

In this study 150 cases with AFI 5cm or less is compared with control group 150 cases with AFI > 5cm.

In the study group – 27 cases of preeclampsia,26 cases of post EDD, 22 cases of previous LSCS,13 cases of breech.

In control group –19 cases of preeclampsia,13 cases of post EDD, 25 cases of previous LSCS,6 cases of breech presentation were taken.

In a study conducted by **Casey & Coworkers B.M.2001** pregnancy outcomes after antepartum diagnosis of oligohydramnios at or beyond 34 weeks gestation in 147 cases. This complication was associated with increase in labour induction(42% vs 18%).Non reassuring heart rate (48% vs 39%).NICU admission (7 % vs 2%), MSAF (1 % vs 0.1%),neonatal death rate (5 % vs 0.3%).

In a study by **Golan & cowokers (1994)** fetal outcome in 145 cases,they found increase incidence of fetal distress, MSAF(29.1%) IUGR(24.5%) Breech 17%.Asphyxia during labour 11.5% , corrected PNMR –10%.

In a study by **Chamberlain & coworkers 1993**,the incidence of major congenital anomaly &IUGR significant related to qualitative AF.

In a study by **Youseef et al 1993** in South Med Journal Measurement of AFI and Fetal outcome was studied in term pregnancies with AFI less than 5 cm and found AFI is superior in detucting fetal outcome.

In study by **Locatelli A 2004** as perinatal outcome associated with oligohydramnios in uncomplicated term pregnancies 40-41.6 weeks .oligohydramnios independently associated with a higher risk of low birth weight percentile.

In study by **Baron C & Coworkers 2000** on impact of AFV on Intrapartum on perinatal outcome.AFI less than or equal to 5 cm compared with Normal.The efficacy of oligohydramnios predicting caesarean delivery for fetal distress gave a sensitivity of 78% a specificity of 74%, +ve predictive value 33% and -ve predictive value 95%.The AFI for

detecting intrapartum oligohydramnios is a valuable screening test for subsequent fetal distress requiring caesarean delivery.

In a study by **Chauhan.SP & coworkers 1999** an antepartum & intrapartum amniotic fluid index of ≤5 cm is associated with a significantly increased risk of CS delivery for fetal distress and low apgar scores at 5mins.

PRESENT STUDY

In this study perinatal outcome in 150 cases AFI ≤ 5 cm and 150 cases of control group are studied. In AFI ≤5 cm, 55 patients had vaginal delivery & 95 patients had LSCS (74PrimaryCS + 21 Rpt LSCS).

In control group about 94 patients had vaginal delivery,56 patients had LSCS(34PrimaryCS + 22 Rpt LSCS)

INDUCTION OF LABOUR:

INDUCTION	**AFI ≤5 cm**	**Control Group**
Present Study	42%	25%
Casey Co Workers (2000)	42%	18%

Induction of labour is comparable with the study by **Casey & coworkers in AmJOG 2000**

In present study 48 cases in AFI ≤ 5 cm & 30 cases in control group were induced.

AFI ≤ 5 cm------ Labour Natural -12

Emg LSCS -36

Control group------Labour Natural -18

Emg LSCS -12

CAESAREAN SECTION FOR FETAL DISTRESS:

Youseef et al (1993)	86.6%
Baron Morgan et al (2000)	78%
Present Study	75%

The efficacy of oligohydramnios predicting CS for Fetal distress has sensitivity of 78% comparable with various studies .

MECONIUM STAINED LIQUOR

Youseef et al (1993)	63.6%
Present Study	64%

The sensitivity of meconium stained liquor is 64% comparable with study.

APGAR SCORE less than 7 @5min.

Youseef et al(1993)	88.8%
Present Study	85%

The sensitivity of oligohydraminos in predicting apgar less than 7 @ 5 min is comparable.

IUGR

Youseef et al (1993)	79.9%
Present Study	79%

The sensitivity of AFI measurement of $\leq$ 5 cm is comparable with study.

PERINATAL MORTALITY

(American Journal of Obs & Gyn. 2000)

Neonatal	**AFI ≤ 5 cm**	**Control Group**
Present Study	6%	0.3%
Casey Co Workers (2000)	5%	0.3%

Neonatal death rate is comparable with study.

NEONATAL DEATH

- In study group there were 9 neonatal deaths out of this 8 were due to complications of IUGR, In control group 1 neonatal death.

- **Case No – 15**.Maheswari 23F Primi. Unbooked, GA – 41 weeks 3 days on admission with NR –NST,AFI 3.4 cm ,with unfavourable cervix. Emergency LSCS done indication IUGR with fetal distress. Alive boy, 1.75 kg apgar 7/10, 8/10,thick meconium, admitted in NICU. Term IUGR Baby had NNEC, meningitis , Jaundice , died after one week .

- **Case No – 19**.Amsavalli 19F Primi Unbooked, GA – 38 weeks 4 days, breech presentation,AFI- 3 cm, NST-NR.Emergency LSCS done immediately in view of severe oligohydramnios and breech presentation. indication IUGR. breech with fetal distress. Alive boy,1.65 kg apgar 6/10,7/10,thin meconium admitted in NICU. Baby died after two days due to RDS.

- **Case No-22**.zeenath 24F Primi. Booked, GA – 38 weeks on admission NR NST,AFI – 4, with thick meconium in early labour taken for Emergency LSCS within 30 min.Alive boy 1.7 kg 6/10.7/10. thick Meconium. IUGR admitted in NICU died after one day due to MAS

- **Case No – 58** .Rajakumari 24F Primi. Unbooked, GA – 39 weeks 3 days NST – variable deceleration AFI - 4 cm, with Unfavourable cervix. Emergency LSCS indication fetal distress. Alive girl – 2.9 kg 6/10.7/10. clear liquor. Admitted in NICU died after 2 days due to RDS.

- **Case No – 86**.Shanthi 23F Primi.Unbooked,GA - 37 weeks 4 days NST – Reactive, AFI – 3 cm .spontaneous labour, labour natural Alive girls 1.5 kg 7/10.8/10. clear liquor. Term IUGR Admitted in NICU. Baby had recurrent seizures, meningitis,NNEC died after 5 days.

- **Case No – 91**.Regina. 24F Primi. Booked, Preeclampsia.GA – 38 weeks 5 days NST – reactive, Unfavourable cervix . Induced with PGE 2 gel twice not responded.Emergency LSCS indication IUGR with failed induction. Alive boy 1.6kg, 6/10.8/10. thin meconium. Term IUGR admitted in NICU. Baby had Polycythemia and died after 2 days due to Pulmonary haemorrhage.

- **Case No – 98**.Narasamma.32F G2 A1 Md 8 Yrs Unbooked GA – 39 weeks NST – NR ,AFI - <2 . Doppler High resistance flow, transverse lie.In View of severe Oligo hydramnios transverse lie, long period of infertility Emergency LSCS done Alive Girl – 1.4kg , 3/10.6/10 . thick meconium admitted in NICU. Baby died after 3 days due to perinatal asphyxia.

- **Case No – 113**.Hemalatha.22F G2 P1 L1 Prev LSCS, Booked GA – 39 weeks AFI – 2 .Admitted with decreased fetal movement NST – NR, with Unfavourable cervix .Emergency repeat LSCS done Alive boy – 2.6 kg.2/10.3/10.thick meconium term IUGR admitted in NICU. Baby died after 2 hours due to MAS.

- **Case No – 118.**Kiruparani.23F G3 A2 . Md 4 Yrs Booked GA – 39weeks AFI- 2 NST – NR with Unfavourable cervix Emergency LSCS done indication severe Oligohydramnios with IUGR.Alive Boy – 1.8kg 7/10.8/10. thick meconium, Term IUGR ,PN hypoxia,HIE III Admitted in NICU . Baby died after 2 days

- **In control group**

Case No – 83.Dhanalakshmi 25F G1 severe Preeclampsia, Unbooked GA 39 weeks, AFI 8.9cm, NST – NR,Doppler MCA Increase diastolic flow with Unfavourable cervix. Induced with PGE2 gel Not – responded Emerancy LSCS done indication severe PET IUGR with fetal distress.Alive girl 1.6kg,3/10.4/10.thin meconium Term IUGR HIE III Admitted in NICU. Baby discharged after 7 days, died 8 days after discharge at home.

SUMMARY

In this study perinatal outcome in AFI 5cm or less compared with control group.

About 150 cases were studied in each group.

- 87% in study group & 93% in control group were in the age group 20 - 30yrs.
- In study group 37% had vaginal & 63% has LSCS(22% were repeat LSCS). In control group 63% had vaginal & 37% had LSCS delivery (39% were Repeat LSCS)
- The risk of CS for fetal distress found to be higher in study group than control (32 Vs 11%,) The difference was found to be significant (P=0.05).
- In study group, induction was found to be higher than control (42% Vs 25%). The difference was found to be significant. (P=0.003)
- Meconium stained liquor was 46% in study group, 25% in control group. The difference was not significant (P=0.17).
- In study group 11% had apgar score <7 @ 5 min Vs 1% in control group.The difference was found to be significant. (P=0.001)
- In study group 18% of babies were less than 2 kg, 5% in control group were less than 2 kg. The difference was found to be significant. (P=0.001).

- In study group about 67% had reactive NST & 33% had NR NST. In Control group 84% had Reactive NST, 16% had Non reactive NST. (P=0.002)
- In Oligohydramnios (Study Group)

  The risk of apgar less than 7 at 5 minutes is high in Non reactive NST (22%) Vs 5% in Reactive NST.

  The risk of NICU admission was found to be high in Non-reactive NST (65%) Vs 37% in Reactive NST. The difference was found to be significant. (P= 0.002).

  Neonatal death is 12% in Non-reactive NST and 3% in Reactive NST. The difference was found to be significant. (P = 0.02).

In control group - the risk of apgar less than 7@ 5 min is only 8% in Non reactive NST and 0.8 in reactive NST.

The risk of NICU admission is 38% in Non-Reactive NST, 13% in Reactive NST.The difference was found to be significant.(P=0.02).

CONCLUSION

- Oligohydramnios is associated with adverse perinatal outcome.

- Oligohydramnios with reactive NST is associated with good prognosis (good apgar, decreased NICU admission & neonatal death).

- Oligohydramnios with non reactive NST needs careful monitoring and eventuates in early delivery. It increases the incidence of caesarean delivery for fetal distress, NICU admission, low apgar at 5 mins and Neonatal death.

- Oligohydramnios associated with IUGR carries a poor perinatal outcome (increased neonatal death, NICU admission, increased rate of CS for fetal distress, very low birth weight) Hence they need good neonatal care.

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PROFORMA

Name Age Unit IP No. G P L A

Address Md Since

Booked / Unbooked DOA

Menstural History LMP –

EDD –

Maternal Complication

G.E – Anemia / Pedal Edema / Temp

Obstetric Examination

Ht of fundus –

Presentation –

FHR -

Liquor clinically - Adequate / Not Adequate

Investigations:

Urine - Albumin Hb % - Other investigations

Sugar Blood group

Deposits

USG – GA –

Placenta NST – Reactive / Non reactive

FH

AFI

Mode of Delivery

Spontaneous ☐ Induction ☐

Vaginal ☐ Instrumental ☐ LSCS - EMG/Elective

Induction Delivery Interval

Drip given / not

FHR Variation

Duration of Labour –

Rupture of Memb –

Colour of Liquor –

Baby Details:

Cried after birth - Yes / No

Sex - Boy / Girl

Wt -

Apgar -

Baby Resucitated -

Admission in NICU - Yes / No Days –

MSAF - Yes / No

Associated Complication

for baby -

Follow up:

Whether Baby discharged in good condition.

28 Days follow up –

EXPANSION OF ABBREVIATIONS

AF	**-**	**Amniotic Fluid**
AFI	**-**	**Amniotic Fluid Index**
AFV	**-**	**Amniotic Fluid Volume**
H	**-**	**Foetal Heart**
GA	**-**	**Gestational Age**
LSCS	**-**	**Lower Segment Caesarean Section**
NST	**-**	**Non Stress Test**